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FILE 'HOME' ENTERED AT 16:12:22 ON 25 JUL 2007

=> index bioscience

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FULL ESTIMATED COST

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67 FILES IN THE FILE LIST IN STNINDEX

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=> decl and ppar

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9	FILE CAPLUS
88	FILE DGENE
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1	FILE EMBASE
1	FILE ESBIOBASE
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14	FILE USPATFULL
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16 FILES HAVE ONE OR MORE ANSWERS, 67 FILES SEARCHED IN STNINDEX

L1 QUE DECL AND PPAR

=> d rank

F1	88	DGENE
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F3	9	CAPLUS
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F5	2	IFIPAT
F6	2	WPIDS
F7	2	WPINDEX
F8	1	BIOTECHABS
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F11	1	EMBASE
F12	1	ESBIOBASE
F13	1	LIFESCI
F14	1	MEDLINE
F15	1	SCISEARCH
F16	1	TOXCENTER

=> fule caplus biosis ifipat biotechabs

43 FILES SEARCHED...

0 FILES HAVE ONE OR MORE ANSWERS, 67 FILES SEARCHED IN STNINDEX

L2 QUE FULE CAPLUS BIOSIS IFIPAT BIOTECHABS

=> medline

1293	FILE ADISCTI
2	FILE ADISINSIGHT
4395	FILE ADISNEWS
140	FILE AGRICOLA
7	FILE ANABSTR
11	FILE ANTE
2	FILE AQUALINE
6	FILE AQUASCI
154	FILE BIOENG
9907	FILE BIOSIS
44	FILE BIOTECHABS
44	FILE BIOTECHDS
659	FILE BIOTECHNO
922	FILE CABA
3998	FILE CAPLUS
14	FILE CEABA-VTB
4	FILE CIN
37	FILE CONFSCI
2	FILE CROPU
112	FILE DDFU
95	FILE DISSABS
2	FILE DRUGMONOG2
419	FILE DRUGU
642	FILE EMBAL
22950	FILE EMBASE
4847	FILE ESBIODBASE
13	FILE FROSTI
37	FILE FSTA
24	FILE GENBANK
255	FILE HEALSAFE
24	FILE IFIPAT
11	FILE KOSMET
764	FILE LIFESCI
24172	FILE MEDLINE
307	FILE NTIS
3	FILE NUTRACEUT
3	FILE OCEAN
10409	FILE PASCAL
9	FILE PHARMAML
94	FILE PHIN
1507	FILE PROMT
3	FILE RDISCLOSURE
16489	FILE SCISEARCH
8409	FILE TOXCENTER
817	FILE USPATFULL
78	FILE USPAT2
1	FILE WATER
18	FILE WPIDS
65	FILES SEARCHED...
18	FILE WPINDEX

49 FILES HAVE ONE OR MORE ANSWERS, 67 FILES SEARCHED IN STNINDEX

L3 QUE MEDLINE

=> file	caplus	biosis	ifipat	biotechabs	medline	scisearch	toxcenter	
COST IN U.S. DOLLARS					SINCE FILE		TOTAL	

	ENTRY	SESSION
FULL ESTIMATED COST	2.52	2.73

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=> decl and ppar
L4 16 DEC1 AND PPAR

=> dup remove
ENTER L# LIST OR (END):l4
PROCESSING COMPLETED FOR L4
L5 12 DUP REMOVE L4 (4 DUPLICATES REMOVED)

=> d ti 1-12

L5 ANSWER 1 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
TI Gene expression profiling in peripheral blood mononuclear cells in the
diagnosis and therapy of vascular disease

L5 ANSWER 2 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
TI Test method and kit for examining balance of Th1 helper T cells and Th2
helper T cells

L5 ANSWER 3 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
TI Gene expression profiles in the diagnosis and treatment of Alzheimer's
disease

L5 ANSWER 4 OF 12 IFIPAT COPYRIGHT 2007 IFI on STN
TI METHODS AND COMPOSITIONS FOR REGULATING ADIPOGENESIS

L5 ANSWER 5 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
TI Hypoxia inhibits adipocyte differentiation in a HDAC-independent manner

L5 ANSWER 6 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
TI Mouse genes differentially expressed in Th1 or Th2 cells for use in
determining Th1/Th2 balance

L5 ANSWER 7 OF 12 IFIPAT COPYRIGHT 2007 IFI on STN
TI FAT REGULATION

L5 ANSWER 8 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
TI Oxygen-dependent regulation of adipogenesis

L5 ANSWER 9 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 1
TI Sequence of truncated transcription factor DEC1/Stral3 from

human and mouse and their uses in adipogenesis and angiogenesis inhibition

L5 ANSWER 10 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
TI CREB controls hepatic lipid metabolism through nuclear hormone receptor
PPAR- γ

L5 ANSWER 11 OF 12 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on
STN

TI Inhibition of PPARgamma2 gene expression by the HIF-1 regulated gene
DEC1/Stra13: A mechanism for regulation of differentiation by
hypoxia.

L5 ANSWER 12 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 2
TI Inhibition of PPAR.gamma.2 gene expression by the
HIF-1-regulated gene DEC1/Stra13: a mechanism for regulation of
adipogenesis by hypoxia

=> d ab bib 12, 11, 10, 9, 8, 7, 5, 4

L5 ANSWER 12 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 2
AB Cellular differentiation involves transcriptional responses to
environmental stimuli. Adipocyte differentiation is inhibited under
hypoxic conditions, indicating that oxygen (O₂) is an important physiol.
regulator of adipogenesis. Hypoxia inhibits PPAR.gamma.2
nuclear hormone receptor transcription, and overexpression of PPAR
 γ 2 or C/EBP β stimulates adipogenesis under hypoxia. Mouse
embryonic fibroblasts deficient in hypoxia-inducible transcription factor
1 α (HIF-1 α) are refractory to hypoxia-mediated inhibition of
adipogenesis. The HIF-1-regulated gene DEC1/Stra13, a member of
the Drosophila hairy/Enhancer of split transcription repressor family,
represses PPAR.gamma.2 promoter activation and functions as an
effector of hypoxia-mediated inhibition of adipogenesis. These data
indicate that an O₂-sensitive signaling mechanism regulates adipogenesis.
Thus, agents that regulate HIF-1 activity or O₂ sensing may be used to
inhibit adipogenesis and control obesity.

AN 2002:225678 CAPLUS <<LOGINID::20070725>>

DN 136:338236

TI Inhibition of PPAR.gamma.2 gene expression by the
HIF-1-regulated gene DEC1/Stra13: a mechanism for regulation of
adipogenesis by hypoxia

AU Yun, Zhong; Maecker, Heather L.; Johnson, Randall S.; Giaccia, Amato J.
CS Department of Radiation Oncology, Stanford University, Stanford, CA,
94305, USA

SO Developmental Cell (2002), 2(3), 331-341
CODEN: DCEEBE; ISSN: 1534-5807

PB Cell Press

DT Journal

LA English

RE.CNT 71 THERE ARE 71 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 11 OF 12 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on
STN

AN 2002:419264 BIOSIS <<LOGINID::20070725>>

DN PREV200200419264

TI Inhibition of PPARgamma2 gene expression by the HIF-1 regulated gene
DEC1/Stra13: A mechanism for regulation of differentiation by
hypoxia.

AU Yun, Zhong [Reprint author]; Maecker, Heather L.; Johnson, Randall S.;
Giaccia, Amato J.

CS Stanford University School of Medicine, Stanford, CA, USA

SO Proceedings of the American Association for Cancer Research Annual
Meeting, (March, 2002) Vol. 43, pp. 1024. print.

Meeting Info.: 93rd Annual Meeting of the American Association for Cancer Research. San Francisco, California, USA. April 06-10, 2002.
ISSN: 0197-016X.

DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 7 Aug 2002
Last Updated on STN: 7 Aug 2002

L5 ANSWER 10 OF 12 CAPLUS. COPYRIGHT 2007 ACS on STN

AB Fasting triggers a series of hormonal cues that promote energy balance by inducing glucose output and lipid breakdown in the liver. In response to pancreatic glucagon and adrenal cortisol, the cAMP-responsive transcription factor CREB activates gluconeogenic and fatty acid oxidation programs by stimulating expression of the nuclear hormone receptor coactivator PGC-1 (refs. 2-5). In parallel, fasting also suppresses lipid storage and synthesis (lipogenic) pathways, but the underlying mechanism is unknown. Mice deficient in CREB activity have a fatty liver phenotype and display elevated expression of the nuclear hormone receptor PPAR- γ , a key regulator of lipogenic genes. CREB inhibits hepatic PPAR- γ expression in the fasted state by stimulating the expression of the Hairy Enhancer of Split (HES-1) gene, a transcriptional repressor that is shown here to be a mediator of fasting lipid metabolism in vivo. The coordinate induction of PGC-1 and repression of PPAR- γ by CREB during fasting provides a mol. rationale for the antagonism between insulin and counter-regulatory hormones, and indicates a potential role for CREB antagonists as therapeutic agents in enhancing insulin sensitivity in the liver.

AN 2003:886576 CAPLUS <<LOGINID::20070725>>

DN 140:105796

TI CREB controls hepatic lipid metabolism through nuclear hormone receptor PPAR- γ

AU Herzig, Stephan; Hedrick, Susan; Morantte, Ianessa; Koo, Seung-Hoi; Galimi, Francesco; Montminy, Marc

CS Peptide Biology Laboratories Salk Institute for Biological Studies, La Jolla, CA, 92037-1002, USA

SO Nature (London, United Kingdom) (2003), 426(6963), 190-193
CODEN: NATUAS; ISSN: 0028-0836

PB Nature Publishing Group

DT Journal

LA English

RE.CNT 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 9 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 1

AB The invention provides protein sequences of truncated transcription factor DEC1/Stral3 from human and mouse, which lacks the DEC1/Stral3 repressor domain. The disclosure provides compns. comprising one or more DEC1/Stral3 fragments of capable of inhibiting PPAR. γ 2 promoter activity. These fragments, e.g. the basic helix loop helix domain or amino acids 1-141, have substantially the same PPAR γ 2 promoter repressing activity as the full length polypeptide. The present disclosure relates to methods and compns. for hypoxia-mediated adipogenic inhibition. The present disclosure provides methods of inhibiting adipogenesis comprising contacting a cell with a fragment of DEC1/Stral3. The invention further relates to methods and compns. of inhibiting angiogenesis in a tumor comprising contacting a tumor or tumor cell with a DEC1/Stral3 agonist.

AN 2003:696706 CAPLUS <<LOGINID::20070725>>

DN 139:225520

TI Sequence of truncated transcription factor DEC1/Stral3 from human and mouse and their uses in adipogenesis and angiogenesis inhibition

IN Giaccia, Amato J.; Yun, Zhong

PA The Board of Trustees of the Leland Stanford Junior University, USA

SO PCT Int. Appl., 49 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2003072064	A2	20030904	WO 2003-US6360	20030228
	W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW			
	RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			
	AU 2003213663	A1	20030909	AU 2003-213663	20030228
	US 2005282167	A1	20051222	US 2005-524919	20050217
PRAI	US 2002-360689P	P	20020228		
	WO 2003-US6360	W	20030228		

L5 ANSWER 8 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
AB Hypoxia is a physiolo. regulator of adipogenesis. Nevertheless, it can potentially play a significant role in the differentiation of many other cell types. The effects of hypoxia on oxygen-dependent regulation of PPAR, C/EBP and DEC1/Stral3 gene expression and adipogenesis are presented. The regulation of adipogenesis by hypoxia serves as a model for future research in understanding how the cellular microenvironment regulates cell differentiation during both embryogenesis and development of adult stem cells.
AN 2004:403942 CAPLUS <<LOGINID::20070725>>
DN 141:409025
TI Oxygen-dependent regulation of adipogenesis
AU Swiersz, Lillian M.; Giaccia, Amato J.; Yun, Zhong
CS Department of Gynecology and Obstetrics, Stanford University, Stanford, CA, 94305, USA
SO Methods in Enzymology (2004), 381(Oxygen Sensing), 387-395
CODEN: MENZAU; ISSN: 0076-6879
PB Elsevier
DT Journal
LA English
RE.CNT 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 7 OF 12 IFIPAT COPYRIGHT 2007 IFI on STN
AB The present invention provides methods and compounds for regulating fat metabolism and achieving fat homeostasis in a subject. Methods and compound for regulating body weight, reducing body fat, and inducing weight loss are also provided, as are methods and compounds for treating or preventing obesity and for preventing or treating conditions associated with altered fat metabolism including, e.g., obesity, diabetes, atherosclerosis, etc.
AN 10727832 IFIPAT;IFIUDB;IFICDB <<LOGINID::20070725>>
TI FAT REGULATION
INF Fournery; Patrick D., Walnut Creek, CA, US
Guenzler-Pukall; Volkmar, San Leandro, CA, US
Klaus; Stephen J., San Francisco, CA, US
Lin; Al Y., Castro Valley, CA, US
Neff; Thomas B., Atherton, CA, US
Seeley; Todd W., Moraga, CA, US
IN Fournery Patrick D; Guenzler-Pukall Volkmar; Klaus Stephen J; Lin Al Y; Neff Thomas B; Seeley Todd W

PAF Unassigned
PA Unassigned Or Assigned To Individual (68000)
PPA FibroGen Inc (Probable)
AG Intellectual Property Department; FIBROGEN, INC., 225 Gateway Boulevard,
South San Francisco, CA, 94080, US
PI US 2004235082 A1 20041125
AI US 2003-729167 20031204
PRAI US 2002-431351P 20021206 (Provisional)
US 2003-476331P 20030606 (Provisional)
US 2003-476726P 20030606 (Provisional)
FI US 2004235082 20041125
DT Utility; Patent Application - First Publication
FS CHEMICAL
APPLICATION
ED Entered STN: 1 Dec 2004
Last Updated on STN: 30 Nov 2006
PARN This application claims the benefit of U.S. Provisional Application
Serial No. 60/431,351, filed on 6 Dec. 2002; U.S. Provisional Application
Serial No. 60/476,331, filed on 6 Jun. 2003; and U.S. Provisional
Application Serial No. 60/476,726, filed on 6 Jun. 2003, each of which is
incorporated by reference herein in its entirety.
CLMN 33
GI 11 Figure(s).
FIGS. 1A, 1B, and 1C show levels of leptin in human cell culture media
following treatment with various compounds of the invention. Cell lines
shown in the figure are preadipocytes, adipocytes, human foreskin
fibroblasts (HFF), human microvascular endothelial cells (HMEC-1), human
umbilical vein endothelial cells (HUVEC), human hepatocellular carcinoma
cells (Hep 3B), adenovirus-transformed fetal kidney epithelium cells
(293A), and cervical epithelial carcinoma cells (HeLa).
FIGS. 2A and 2B show increase in expression of genes encoding proteins
involved in fat metabolism and distribution in liver of animals treated
with a compound of the invention. FIG. 2A shows expression of various fat
metabolism genes, including apolipoprotein A-IV, acyl CoA thioesterase,
carnitine acetyl transferase, and insulin-like growth factor binding
protein (IGFBP)-1. FIG. 2B shows expression of the plasminogen activator
inhibitor (PAI)-1 gene.
FIGS. 3A, 3B, and 3C show changes in expression of genes encoding factors
involved in cellular response to fatty acids and triglycerides. FIG. 3A
shows changes in expression of DEC1/ Stra13 over time following
treatment with a compound of the invention. FIG. 3B shows increased
expression of DEC1/Stra13 in several tissues following
treatment. FIG. 3C shows decreased expression of peroxisome proliferator
activated receptor (PPAR)gamma following treatment with
compounds of the invention.
FIGS. 4A and 4B show changes in body and organ weight in animals treated
with various doses of a compound of the invention. FIG. 4A shows
dose-dependent retardation in weight gain in animals treated with a
compound of the invention. FIG. 4B shows that the weight loss in animals
is not due to loss of muscle and/or vital organ weight, as exemplified by
the heart.
FIG. 5 shows a dose-dependent reduction in visceral fat in animals treated
with a compound of the invention.
FIGS. 6A, 6B, and 6C show decreased body weight gain and abdominal fat pad
weight in an animal model of diet-induced obesity upon treatment with a
compound of the invention.
FIG. 7 shows decreased serum triglyceride levels in an animal model of
diabetes when treated with a compound of the invention.
FIGS. 8A and 8B show dose-dependent HIF-1 alpha stabilization in cells
treated with compounds of the invention.
FIGS. 9A and 9B show induction of glucose transporter-1 (GluT-1) and
aldolase in cells treated with compounds of the invention.
FIGS. 10A, 10B, and 10C show increase in expression of genes involved in
glucose regulation in the kidney, liver, and lung, respectively, in

animals treated with a compound of the invention.
FIG. 11 shows dose response for oxygen consumption in cervical
adenocarcinoma (HeLa) and transformed fetal kidney (293A) cells treated
with a compound.

L5 ANSWER 5 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
AB Oxygen is the most important factor for the appropriate regulation of
multiple energy homeostasis and cell differentiation. Although
hypoxia-induced signaling cascades have been intensively studied, the mol.
mechanism by which hypoxic signals suppress adipocyte differentiation is
unclear. Here, we demonstrated that repression of adipocyte
differentiation by hypoxia and HIF1 α - or Stral3 -overexpression was
not associated with HDACs. Furthermore, HDACs did not affect inhibitory
effect of Stral3 on PPAR.gamma. promoter activity, although the
hypoxia-induced suppression of adipogenesis was accompanied with reduced
acetylation of histone H3 and H4 at the PPAR.gamma. promoter.
Instead, we revealed that hypoxic circumstances biphasically activated
AMPK and concomitantly blocked clonal expansion of preadipocytes, which is
an indispensable step for early phase of adipocyte differentiation. Taken
together, these results suggest that hypoxic condition attenuates
adipocyte differentiation by inhibition of PPAR.gamma.
expression in a HDAC-independent manner and by activation of AMPK which
impairs clonal expansion phase.
AN 2005:583396 CAPLUS <<LOGINID::20070725>>
DN 143:95175
TI Hypoxia inhibits adipocyte differentiation in a HDAC-independent manner
AU Kim, Kang Ho; Song, Min Jeong; Chung, Jieun; Park, Hyunsung; Kim, Jae Bum
CS Department of Biological Sciences, Seoul National University, Seoul,
151-742, S. Korea
SO Biochemical and Biophysical Research Communications (2005), 333(4),
1178-1184
CODEN: BBRCA9; ISSN: 0006-291X
PB Elsevier
DT Journal
LA English
RE.CNT 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 4 OF 12 IFIPAT COPYRIGHT 2007 IFI on STN
AB According to the disclosure hypoxia-mediated adipogenic inhibition
involves the repression of PPAR gamma 2 expression and its
activity is a common mechanism for adipogenic inhibition by a variety of
stimuli. The present disclosure relates to methods and compositions for
regulating adipogenesis. The disclosure provides compositions comprising
one or more DEC1/Stral3 fragments of capable of inhibiting
PPAR gamma 2 promoter activity. These fragments, e.g. the basic
helix loop helix domain or amino acids 1-141, have substantially the same
PPAR gamma 2 promoter repressing activity as the full length
polypeptide. The present disclosure provides methods of inhibiting
adipogenesis comprising contacting a cell with a fragment of DEC1
/Stral3. The invention further relates to methods and compositions of
inhibiting angiogenesis in a tumor comprising contacting a tumor or tumor
cell with a DEC1/Stral3 agonist.
AN 11043410 IFIPAT;IFIUDB;IFICDB <<LOGINID::20070725>>
TI METHODS AND COMPOSITIONS FOR REGULATING ADIPOGENESIS
INF Giaccia; Amato J, Stanford, CA, US
Yun; Zhong, Redwood city, CA, US
IN Giaccia Amato J; Yun Zhong
PAF Unassigned
PA Unassigned Or Assigned To Individual (68000)
AG BAKER & BOTTS, 30 ROCKEFELLER PLAZA, NEW YORK, NY, 10112, US
PI US 2005282167 A1 20051222
AI US 2003-524919 20030228
WO 2003-US6360 20030228

20050217 PCT 371 date
 20050217 PCT 102(e) date
 PRAI US 2002-360689P 20020228 (Provisional)
 FI US 2005282167 20051222
 DT Utility; Patent Application - First Publication
 FS CHEMICAL APPLICATION
 ED Entered STN: 23 Dec 2005
 Last Updated on STN: 23 Dec 2005
 GOVI This invention was made with government support under National Institutes of Health Grants CA88480 and CA67166 and under National Institutes of Health Cancer Biology Training Grant CA09302. The government has certain rights in the invention.
 CLMN 33
 GI 7 Figure(s).
 FIG. 1. Hypoxia inhibits adipogenesis
 FIG. 2. HIF-1 is required for hypoxia-mediated inhibition of adipogenesis
 MEFs with HIF-1 alpha alleles flanked by loxP sites were incubated with cre-adenovirus (Cre) or control adenovirus (Control) and induced to differentiate as described. CoCl2 or DFO was added at the indicated final concentrations for the entire course of treatment. Cells were stained on Day 7 and photographed (x20).
 FIG. 3. Hypoxia modulates the expression of PPAR gamma 2, C/EBP beta and C/EBP delta
 FIG. 4. Ectopic expression of C/EBP beta or PPAR gamma 2 restores the adipogenic potentials of 3T3-L1 cells under hypoxia.
 FIG. 5. DEC1/Stral3 expression is regulated by O2 tensions via HIF-1
 FIG. 6. DEC1/Stral3 represses PPAR gamma 2 promoter activity
 FIG. 7. Ectopic expression of DEC1/Stral3 inhibits differentiation of 3T3-L1 preadipocytes

=> log y

COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
42.49	45.22

FULL ESTIMATED COST

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE	TOTAL
ENTRY	SESSION
-3.90	-3.90

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